10/647,368

STN Columbus

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* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  SEP 09   CA/CAplus records now contain indexing from 1907 to the
                   present
 NEWS  4  DEC 08   INPADOC: Legal Status data reloaded
 NEWS  5  SEP 29   DISSABS now available on STN
 NEWS  6  OCT 10   PCTFULL: Two new display fields added
 NEWS  7  OCT 21   BIOSIS file reloaded and enhanced
 NEWS  8  OCT 28   BIOSIS file segment of TOXCENTER reloaded and enhanced
 NEWS  9  NOV 24   MSDS-CCOHS file reloaded
 NEWS 10  DEC 08   CABA reloaded with left truncation
 NEWS 11  DEC 08   IMS file names changed
 NEWS 12  DEC 09   Experimental property data collected by CAS now available
                   in REGISTRY
 NEWS 13  DEC 09   STN Entry Date available for display in REGISTRY and CA/CAplus
 NEWS 14  DEC 17   DGENE: Two new display fields added
 NEWS 15  DEC 18   BIOTECHNO no longer updated
 NEWS 16  DEC 19   CROPU no longer updated; subscriber discount no longer
                   available
 NEWS 17  DEC 22   Additional INPI reactions and pre-1907 documents added to CAS
                   databases
 NEWS 18  DEC 22   IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
 NEWS 19  DEC 22   ABI-INFORM now available on STN
 NEWS 20  JAN 27   Source of Registration (SR) information in REGISTRY updated
                   and searchable
 NEWS 21  JAN 27   A new search aid, the Company Name Thesaurus, available in
                   CA/CAplus
 NEWS 22  FEB 05   German (DE) application and patent publication number format
                   changes

 NEWS EXPRESS  DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
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FILE 'HOME' ENTERED AT 12:01:17 ON 27 FEB 2004

=> file caplus, biosis, medline, kosmet
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21
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FILE 'MEDLINE' ENTERED AT 12:01:45 ON 27 FEB 2004

FILE 'KOSMET' ENTERED AT 12:01:45 ON 27 FEB 2004
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=> s 54-11-5/rn
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
L1       16261 54-11-5/RN

=> s l1 and (slimming or cellulite or anticellulite or lipolysis or body fat)
L2          24 L1 AND (SLIMMING OR CELLULITE OR ANTICELLULITE OR LIPOLYSIS OR
               BODY FAT)

=> dup rem l1
DUPLICATE IS NOT AVAILABLE IN 'KOSMET'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING IS APPROXIMATELY   7% COMPLETE FOR L1
PROCESSING IS APPROXIMATELY  15% COMPLETE FOR L1
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PROCESSING IS APPROXIMATELY  78% COMPLETE FOR L1
PROCESSING IS APPROXIMATELY  87% COMPLETE FOR L1
PROCESSING IS APPROXIMATELY  97% COMPLETE FOR L1
PROCESSING COMPLETED FOR L1
L3       16228 DUP REM L1 (33 DUPLICATES REMOVED)

=> dup rem l2
DUPLICATE IS NOT AVAILABLE IN 'KOSMET'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L2
L4          24 DUP REM L2 (0 DUPLICATES REMOVED)

=> d l4 ibib kwic

L4   ANSWER 1 OF 24  CAPLUS  COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER:         2003:991308  CAPLUS
DOCUMENT NUMBER:          140:31505
TITLE:                    Compositions containing angiogenic phospholipids and
                          growth factors and oligosaccharides for the treatment
                          of edematous fibrosclerotic panniculopathy
INVENTOR(S):              Ghisalberti, Carlo
PATENT ASSIGNEE(S):       Brazil
SOURCE:                   PCT Int. Appl., 26 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
```

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003103614	A2	20031218	WO 2003-IB2216	20030610

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: IT 2002-MI1280 A 20020611
OTHER SOURCE(S): MARPAT 140:31505

AB The invention relates to the use of angiogenic agents for the manuf. compns. for the treatment of **cellulite**, better defined by the term, "edematous fibrosclerotic panniculopathy" to elicit the endothelial repair and/or the neovascularization of the **cellulite**-affected area and tissues. The invention also claims a method for treating **cellulite** which comprises the administration of an angiogenic agent, preferably selected from angiogenic phospholipids, hyaluronan oligosaccharides, peptide growth factors and combination, and, optionally, secondary angiogenic agents. Thus, a topical cream contained lysophosphatidic acid/phosphatidic acid 2.6, hyaluronan 1.5, ovalbumin (contg. growth factors) 9.0, fluid paraffin 5.0, cetyl alc. 5.5, petrolatum 5.5, glyceryl monostearate 33, polyoxyethylene 2-octyl dodecyl ether 3.0, glycerin 7.0, dipropylene glycol 20, perfumes and additives and preservatives qs, and water qs to 100 g.

ST edematous fibrosclerotic panniculopathy topical phospholipid; oligosaccharide hyaluronan **cellulite** phospholipid

IT Skin
(**cellulite**; compns. contg. angiogenic phospholipids and growth factors and oligosaccharides for treatment of edematous fibrosclerotic panniculopathy)

IT Adipose tissue
(disease, edematous fibrosclerotic panniculopathy; compns. contg. angiogenic phospholipids and growth factors and oligosaccharides for treatment of **cellulite**)

IT **54-11-5**, Nicotine 58-61-7, Adenosine, biological studies 58-63-9, Inosine 68-94-0, Hypoxanthine 77-06-5 83-46-5, β-Sitosterol 83-86-3, Phytic acid 98-92-0, Nicotinamide 112-84-5, Erucamide 464-92-6, Asiatic acid 472-11-7, Ruscogenin 531-75-9, Esculin 6805-41-0, Escin 7085-55-4, Troxerutin 9030-23-3, Platelet-derived endothelial cell growth factor 16830-15-2, Asiaticoside 18449-41-7, Madecassic acid 22002-87-5, Oleoyl lysophosphatidic acid 26993-30-6, Sphingosine 1-phosphate 34540-22-2 62683-29-8, Colony stimulating factor 81627-83-0, Macrophage Colony stimulating factor 83869-56-1, Granulocyte Macrophage Colony stimulating factor 106096-92-8, Acidic fibroblast growth factor 106096-93-9, Basic fibroblast growth factor 115939-25-8, Salvianolic acid B 117346-20-0 127464-60-2, Vascular endothelial growth factor 188417-84-7, Vascular endothelial growth factor C 192662-83-2, Vascular endothelial growth factor B 193363-12-1, Vascular endothelial growth factor D 489395-96-2, Vascular endothelial growth factor A 572921-97-2, Angiogenin
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(compns. contg. angiogenic phospholipids and growth factors and oligosaccharides for treatment of edematous fibrosclerotic panniculopathy)

=> d l4 ibib kwic 2-24

L4 ANSWER 2 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 2001:866948 CAPLUS
DOCUMENT NUMBER: 136:16585
TITLE: Chronic maternal nicotine exposure alters neuronal systems in the arcuate nucleus that regulate feeding behavior in the newborn rhesus macaque
AUTHOR(S): Grove, Kevin L.; Sekhon, Harmonjatinder S.; Brogan, Rebecca S.; Keller, Jennifer A.; Smith, M. Susan; Spindel, Eliot R.
CORPORATE SOURCE: Division of Neuroscience, Oregon Regional Primate Research Center, Oregon Health and Science University, Beaverton, OR, 97006, USA
SOURCE: Journal of Clinical Endocrinology and Metabolism (2001), 86(11), 5420-5426
CODEN: JCEMAZ; ISSN: 0021-972X
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB It is well known that maternal smoking during pregnancy can lead to low birth wt. and low **body fat** in human newborns. The purpose of this study was to det. whether chronic maternal nicotine treatment alters the levels of known regulators of energy balance in the newborn offspring. Pregnant rhesus monkeys were treated with nicotine tartrate (1.5 mg/kg·d) starting on d 26 of pregnancy and maintained through d 160 of gestation. Nicotine exposure had no significant effect on abs. birth wts. of the neonatal monkeys, although there was a 10% redn. in birth wts. with nicotine exposure when they were normalized to maternal wt. Postnatal d 1 plasma leptin levels were significantly reduced by ~50% in the nicotine treatment group compared with saline controls, suggesting that the infant monkeys exposed to nicotine may also have lower **body fat** levels. In situ hybridization studies demonstrated that chronic nicotine exposure resulted in a significant decrease in arcuate NPY mRNA expression in the neonatal monkeys. In addn., there was a 2-fold increase in POMC mRNA in the arcuate nucleus in the nicotine-exposed group. These data suggest that nicotine exposure during pregnancy may increase energy expenditure in the developing fetus through actions on hypothalamic systems, resulting in lower birth wts. and **body fat** levels.

IT **54-11-5**, Nicotine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(chronic maternal nicotine exposure alters neuronal systems in arcuate nucleus that regulate feeding behavior in newborn rhesus macaque)

L4 ANSWER 3 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 2001:619077 CAPLUS
DOCUMENT NUMBER: 136:319173
TITLE: Systemic nicotine stimulates human adipose tissue **lipolysis** through local cholinergic and catecholaminergic receptors
AUTHOR(S): Andersson, K.; Arner, P.
CORPORATE SOURCE: Departments of Medicine and Research Center, Huddinge Hospital, Huddinge, Swed.

SOURCE: International Journal of Obesity (2001), 25(8), 1225-1232
CODEN: IJOBDP; ISSN: 0307-0565
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Systemic nicotine stimulates human adipose tissue **lipolysis** through local cholinergic and catecholaminergic receptors

AB OBJECTIVE: To evaluate whether the lipolytic effects of systemic nicotine are not only attributed to indirect adrenergic mechanisms, but also to a direct action of nicotine on fat cells. DESIGN: The effect of a systemic nicotine infusion (0.5 μg/kg/min for 30 min) on **lipolysis** in s.c. adipose tissue was investigated in situ in 11 non-obese, non-smoking, healthy male subjects under placebo-controlled conditions. MEASUREMENTS: By using microdialysis probes the glycerol levels (**lipolysis** index) and blood flow were monitored locally in s.c. adipose tissue. RESULTS: Blood plasma nicotine levels peaked (7.2 ng/mL) at the end of the infusion. Nicotine induced a mean percentage peak increase in adrenaline and noradrenaline plasma levels of 213% and 118%, resp. Nicotine increased venous plasma glycerol levels by 144%, arterialized plasma glycerol levels by 148%, and adipose glycerol levels by 148%, but did not alter blood flow. By inducing a local cholinoceptor blockade with mecamylamine (10-5 M) via the microdialysis system, the increase in adipose glycerol levels was inhibited by ~45%. A corresponding local β-adrenoceptor blockade with propranolol (10-4 M), inhibited the increase in adipose glycerol levels by ~60%. Infusion of saline (ie placebo) had no effect on the parameters mentioned above. CONCLUSION: Systemically administered nicotine induces **lipolysis**, in part by activating the classical adrenergic mechanism (mediated by a nicotine-induced release of catecholamines stimulating β-adrenoceptors), and in part by directly activating a nicotinic cholinergic lipolytic receptor located in adipose tissue.

ST nicotine adipose tissue **lipolysis** nicotinic receptor

IT Lipids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**lipolysis**; systemic nicotine stimulates adipose tissue **lipolysis**)

IT Adipose tissue
Heart rate
Human
(systemic nicotine stimulates adipose tissue **lipolysis**)

IT Nicotinic receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(systemic nicotine stimulates adipose tissue **lipolysis**)

IT Blood pressure
(systolic; systemic nicotine stimulates adipose tissue **lipolysis**)

IT Adrenoceptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β-; systemic nicotine stimulates adipose tissue **lipolysis**)

IT 51-41-2, Noradrenaline 51-43-4, Adrenaline 56-81-5, Glycerol, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(systemic nicotine stimulates adipose tissue **lipolysis**)

IT 486-56-6, Cotinine
RL: BSU (Biological study, unclassified); PKT (Pharmacokinetics); BIOL (Biological study)
(systemic nicotine stimulates adipose tissue **lipolysis**)

IT **54-11-5**, Nicotine

RL: DMA (Drug mechanism of action); PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(systemic nicotine stimulates adipose tissue **lipolysis**)

L4 ANSWER 4 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1999:169485 CAPLUS
DOCUMENT NUMBER: 130:348434
TITLE: Leptin levels in smokers and long-term users of nicotine gum
AUTHOR(S): Eliasson, B.; Smith, U.
CORPORATE SOURCE: Lundberg Laboratory for Diabetes Research, Sahlgrenska University Hospital, Goteborg, S-413 45, Swed.
SOURCE: European Journal of Clinical Investigation (1999), 29(2), 145-152
CODEN: EJCIB8; ISSN: 0014-2972
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB The aim of this study was to examine the effects of cigarette smoking and other forms of long-term nicotine consumption on circulating leptin levels as well as the relationship between leptin levels and insulin sensitivity, measured with the euglycemic hyperinsulinemic clamp, in healthy middle-aged men. Samples from 73 subjects were analyzed: 23 non-smokers, 31 smokers and 19 long-term nicotine gum chewers (NGCs) with similar ranges of age, body mass index (BMI) and per cent **body fat**. Leptin levels were higher in NGCs and smokers than in the non-smoking matched control subjects. Smoking cessation for 8 wk further increased the leptin levels, probably due to the concomitant increase in **body fat** (mean ± SD, 2·2±1·8 kg). Acute administration of one dose of nicotine nasal spray or smoking one cigarette did not significantly change the circulating leptin levels during the following 60 min. Plasma leptin concns. were pos. correlated with the proportion of **body fat** and neg. correlated with the degree of insulin sensitivity in each of the three subject groups. In a stepwise multiple linear regression anal., plasma leptin concns. were significantly correlated with the proportion of **body fat**, degree of insulin sensitivity and smoking status. These data show that long-term use of nicotine is assocd. with elevated circulating leptin levels. The increased leptin levels may be an important reason for the lower body wt. in smokers. The results of this study also support the view that leptin is directly or indirectly related to insulin sensitivity in men.

IT **54-11-5**, Nicotine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(leptin levels in smokers and long-term users of nicotine gum)

L4 ANSWER 5 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1997:406796 CAPLUS
DOCUMENT NUMBER: 127:117296
TITLE: Pharmacodynamics of acute tolerance to multiple nicotinic effects in humans
AUTHOR(S): Fattinger, Karin; Verotta, Davide; Benowitz, Neal L.
CORPORATE SOURCE: Division of Clinical Pharmacology and Experimental Therapeutics, Medical Service, San Francisco General Hospital Medical Center, School of Pharmacy and Deps. of Medicine, Biostatistics and Psychiatry, University of California, San Francisco, CA, USA
SOURCE: Journal of Pharmacology and Experimental Therapeutics (1997), 281(3), 1238-1246

CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: Williams Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Tolerance is an important determinant of addiction as well as therapeutic and/or toxic effects of drugs. The development of acute tolerance to various effects of nicotine was studied in nine healthy smokers who were abstaining from tobacco. Nicotine was infused rapidly to reach a concn. of about 25 ng/mL, followed by a computer-controlled infusion to maintain that concn. A novel semiparametric model of nicotine effects and tolerance was developed. Tolerance to various effects of nicotine (increases in heart rate, blood pressure, plasma epinephrine and energy expenditure) occurred within the range of nicotine levels found in smokers. However, the rate of tolerance development varied considerably. The half-lives of tolerance ranged from 3.5 min for the increase in energy expenditure to 70 min for systolic blood pressure. There was no apparent tolerance to the effects on free fatty acid concns., which reflects **lipolysis**. Differences in the pharmacodynamics of tolerance may reflect differences in rate of desensitization of various subtypes of nicotinic receptors and/or differences in mechanisms of tolerance for various nicotinic effects.

IT Fatty acids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**lipolysis** of; pharmacodynamics of acute tolerance to multiple nicotinic effects in humans)

IT **54-11-5**, Nicotine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(pharmacodynamics of acute tolerance to multiple nicotinic effects in humans)

L4 ANSWER 6 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text

ACCESSION NUMBER: 1997:466983 CAPLUS
DOCUMENT NUMBER: 127:77259
TITLE: No acute effects of smoking and nicotine nasal spray on **lipolysis** measured by subcutaneous microdialysis
AUTHOR(S): Eliasson, B.; Smith, U.; Loennroth, P.
CORPORATE SOURCE: Lundberg Laboratory for Diabetes Research, Sahlgrenska University Hospital, Goteborg, S-413 45, Swed.
SOURCE: European Journal of Clinical Investigation (1997), 27(6), 503-509
CODEN: EJCIB8; ISSN: 0014-2972
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English

TI No acute effects of smoking and nicotine nasal spray on **lipolysis** measured by subcutaneous microdialysis

AB This study investigated adipose tissue **lipolysis** in situ by s.c. microdialysis twice in 10 healthy, male smokers after smoking four cigarettes over 2 h and after the administration of an equal amt. of nicotine given as nasal spray (NNS). Glucose and insulin levels, in situ **lipolysis** and adipose tissue blood flow were studied in the post-absorptive state and after a 75-g oral glucose tolerance test (OGTT). Post-absorptively, acute smoking and NNS increased neither s.c. adipose tissue glycerol prodn. nor plasma free fatty acid (FFA) or glycerol levels. After the OGTT, plasma insulin and lactate levels were significantly higher after smoking, whereas FFA levels were higher after NNS. Normal smoking or the administration of a normal dose of NNS caused only minor metabolic changes. Thus, it does not seem likely that increased **lipolysis** is an important contributor to the dyslipidemia seen

in smokers.
ST tobacco smoking nicotine **lipolysis** adipose tissue
IT Fats and Glyceridic oils, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(lysis of; smoking and nicotine nasal spray effect on **lipolysis** of adipose tissues in humans)
IT Adipose tissue
Blood plasma
Circulation
Tobacco smoke
(smoking and nicotine nasal spray effect on **lipolysis** of adipose tissues in humans)
IT Fatty acids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(smoking and nicotine nasal spray effect on **lipolysis** of adipose tissues in humans)
IT **54-11-5**, Nicotine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(smoking and nicotine nasal spray effect on **lipolysis** of adipose tissues in humans)
IT 50-21-5, biological studies 50-99-7, Glucose, biological studies 51-41-2, Noradrenaline 51-43-4, Adrenaline 56-81-5, Glycerol, biological studies 9004-10-8, Insulin, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(smoking and nicotine nasal spray effect on **lipolysis** of adipose tissues in humans)
IT 486-56-6, Cotinine
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(smoking and nicotine nasal spray effect on **lipolysis** of adipose tissues in humans)

L4 ANSWER 7 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1996:158789 CAPLUS
DOCUMENT NUMBER: 124:223497
TITLE: Alterations of **lipolysis** and lipoprotein lipase in chronically nicotine-treated rats
AUTHOR(S): Sztalryd, Carole; Hamilton, Jock; Horwitz, Barbara A.; Johnson, Patricia; Kraemer, Fredric B.
CORPORATE SOURCE: Sch. Med., Stanford Univ., Stanford, CA, 94305, USA
SOURCE: American Journal of Physiology (1996), 270(2, Pt. 1), E215-E223
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Alterations of **lipolysis** and lipoprotein lipase in chronically nicotine-treated rats
AB These studies examd. the cellular mechanisms for lower adiposity seen with nicotine ingestion. Rats were infused with nicotine or saline for 1 wk and adipocytes isolated from epididymal fat pads. Nicotine-infused rats gained 37% less wt. and had 21% smaller fat pads. Basal **lipolysis** was 78% higher, whereas the maximal lipolytic response to isoproterenol was blunted in adipocytes from nicotine-infused rats. The antilipolytic actions of adenosine and the levels of serum catecholamine were unaffected by nicotine. The nicotine-induced alteration in **lipolysis** was not assocd. with any changes in hormone-sensitive lipase. Nicotine caused a 30% decrease in lipoprotein lipase (LPL) activity, without any changes in

LPL mass or mRNA levels, in epididymal fat in the fed state. In contrast, LPL activity, mass, and mRNA levels in heart were increased by nicotine whether animals were fed or fasted. These studies provide evidence for multiple mechanistic events underling nicotine-induced alterations in wt. and suggest that nicotine diverts fat storage away from adipose tissue and toward utilization by muscle.

ST **lipolysis** lipoprotein lipase nicotine

IT Lipids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**lipolysis** and lipoprotein lipase response to nicotine)

IT Adipose tissue
(adipocyte, **lipolysis** and lipoprotein lipase response to nicotine)

IT **54-11-5**, Nicotine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(**lipolysis** and lipoprotein lipase response to nicotine)

IT 9004-02-8, Lipoprotein lipase
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**lipolysis** and lipoprotein lipase response to nicotine)

L4 ANSWER 8 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text

ACCESSION NUMBER:	1995:758205 CAPLUS
DOCUMENT NUMBER:	123:194966
TITLE:	Cholinoceptor-mediated effects on glycerol output from human adipose tissue using in situ microdialysis
AUTHOR(S):	Andersson, Kurt; Arner, Peter
CORPORATE SOURCE:	Huddinge Hospital, Karolinska Inst., Huddinge, S-14186, Swed.
SOURCE:	British Journal of Pharmacology (1995), 115(7), 1155-62 CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER:	Macmillan Scientific Medical Division
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB Possible cholinoceptor-mediated effects on **lipolysis** were investigated in vivo in human s.c. adipose tissue of non-obese, non-smoking, healthy subjects, by use of microdialysis. Cholinomimetic and sympathomimetic agents were added to the in-going dialyzate solvent. Addn. of nicotine to the perfusion solvent caused a concn.-dependent reversible increase in the levels of glycerol in the dialyzate (**lipolysis** index). The opposite effect (also concn.-dependent and reversible) was caused by the addn. of carbachol. The max. effects were 100% stimulation and 50% inhibition, resp., by nicotine and carbachol. Neither nicotine nor carbachol stimulated nutritive blood flow in adipose tissue (as measured with an ethanol escape technique). The nicotine effect in situ was concn.-dependently counteracted by the nicotinic cholinoceptor antagonist, mecamylamine. Likewise, the carbachol effect was concn.-dependently counteracted by the muscarinic cholinoceptor antagonist, atropine. When adipose tissue was pretreated with phentolamine plus propranolol to obtain a complete α and β-adrenoceptor blockade, and the subsequent addn. of nicotine or carbachol still induced an increase and decrease in dialyzate glycerol levels (lipolytic or antilipolytic effects), resp. When adipose tissue was pretreated with mecamylamine or atropine, the subsequent addn. of acetylcholine caused a reversible decrease and increase, resp., of the dialyzate glycerol levels. Nicotine and carbachol had no effects on glycerol release from human isolated s.c. fat cells that were incubated in vivo. In conclusion, the data demonstrate a dual effect of the cholinoceptor system on glycerol output in human adipose tissue: stimulation through nicotinic receptors and inhibition through muscarinic

receptors. These effects, which are not obsd. in vitro, are independent of the adrenergic system and the local blood flow and seem not to be mediated by a direct action on the fat cell.

IT Lipids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**lipolysis**; cholinoceptor-mediated effects on glycerol output from human adipose tissue using in situ microdialysis)

IT 51-83-2, Carbachol **54-11-5**, Nicotine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(cholinoceptor-mediated effects on glycerol output from human adipose tissue using in situ microdialysis)

L4 ANSWER 9 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text

ACCESSION NUMBER: 1995:577942 CAPLUS
DOCUMENT NUMBER: 122:308579
TITLE: Influence of aerobic fitness, activity level, and smoking history on the acute thermic effect of nicotine
AUTHOR(S): Perkins, Kenneth A.; Sexton, Joan E.
CORPORATE SOURCE: Sch. Med., Univ. Pittsburgh, Pittsburgh, PA, 15213, USA
SOURCE: Physiology Behavior (1995), 57(6), 1097-102
CODEN: PHBHA4; ISSN: 0031-9384
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

AB This study examd. the influence of smoking history, body compn., and, in a subset of subjects, aerobic fitness and phys. activity on the thermic effect of nicotine using a measured-dose nasal spray procedure. Following overnight fasting and smoking abstinence, 38 healthy male smokers were intermittently administered a nicotine dose cor. for body wt. (15 μg/kg, approx. 1.1 mg for av. subject) or placebo on sep. occasions in a within-subjects study. Indirect calorimetry was used to assess resting energy expenditure (REE) before and after dosing. Acute thermic response to nicotine ranged from -4.3 to +10.8 kcal/h (-5.4% to +12.6% of REE). Thermic response to nicotine was correlated significantly with aerobic fitness (r = 0.58), phys. activity (r = 0.44), and no. of pack-years of smoking (r = 0.43). Thermic response was marginally correlated with percent **body fat** (r = 0.23), but not with body wt. (r = -0.04), percent of ideal wt. for height (r = -0.10), or lean body mass (r = 0.05). These results indicate that male smokers higher in fitness and activity and with greater smoking exposure histories may experience greater increases in energy expenditure as a result of nicotine intake via smoking. Consequently, variability in these characteristics could help account for some of the variability in wt. gain after stopping smoking.

IT **54-11-5**, Nicotine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(aerobic fitness and activity level and smoking history effect on the acute thermic effect of nicotine in human)

L4 ANSWER 10 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text

ACCESSION NUMBER: 1994:238073 CAPLUS
DOCUMENT NUMBER: 120:238073
TITLE: Effects of cigarette smoking and its cessation on lipid metabolism and energy expenditure in heavy smokers
AUTHOR(S): Hellerstein, Marc K.; Benowitz, Neil L.; Neese,

Richard A.; Schwartz, Jean-Marc; Hoh, Rebeccah; Jacob, Peyton, III; Hsieh, James; Faix, Dennis
CORPORATE SOURCE: San Francisco Gen. Hosp., Univ. California, San Francisco, CA, 94110, USA
SOURCE: Journal of Clinical Investigation (1994), 93(1), 265-72
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The relationship between thermogenic and potentially atherogenic effects of cigarette smoking (CS) and its cessation was investigated. Heavy smokers (n = 7, serum cotinine > 200 ng/mL, > 20 cigarettes/d) were maintained on isoenergetic, const. diets for 2 wk, 1 wk with and 1 wk without CS. Stable isotope infusions with indirect calorimetry were performed on day 7 of each phase, after an overnight fast. CS after overnight abstention increased resting energy expenditure by 5% (not significant vs. non-CS phase; P = 0.18). CS increased the flux of FFA by 77%, flux of glycerol by 82%, and serum FFA concns. by 73% (P < 0.02 for each), but did not significantly affect fat oxidn. Hepatic reesterification of FFA increased more than threefold (P < 0.03) and adipocyte recycling increased nonsignificantly (P = 0.10). CS-induced lipid substrate cycles represented only 15% (estd. 11 kcal/d) of obsd. changes in energy expenditure. De novo hepatic lipogenesis was low (<1-2 g/d) and unaffected by either acute CS or its chronic cessation. Hepatic glucose prodn. was not affected by CS, despite increased serum glycerol and FFA fluxes. Cessation of CS caused no rebound effects on basal metabolic fluxes. In conclusion, a metabolic mechanism for the atherogenic effects of CS on serum lipids (increased hepatic reesterification of FFA) has been documented. Increased entry of FFA accounts for CS-induced increases in serum FFA concns. The thermogenic effect of CS is small or absent in heavy smokers while the potentially atherogenic effect is maintained, and cessation of CS does not induce a rebound lipogenic milieu that specifically favors accrual of **body fat** in the absence of increased food intake.

IT **54-11-5**, Nicotine 56-81-5, Glycerol, biological studies
486-56-6, Cotinine 9004-10-8, Insulin, biological studies
RL: BIOL (Biological study)
(of blood serum, lipid metab. and energy expenditure in heavy human smokers in relation to)

L4 ANSWER 11 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1990:436279 CAPLUS
DOCUMENT NUMBER: 113:36279
TITLE: Effects of nicotine on body weight, food consumption and body composition in male rats
AUTHOR(S): Winders, Suzan E.; Grunberg, Neil E.
CORPORATE SOURCE: Dep. Med. Psychol., Uniformed Serv. Univ. Health Sci., Bethesda, MD, 20814-4799, USA
SOURCE: Life Sciences (1990), 46(21), 1523-30
CODEN: LIFSAK; ISSN: 0024-3205
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Administration of nicotine was assocd. with attenuated body wt. gains and cessation was assocd. with accelerated body wt. gains. Changes in fat compn. paralleled changes in body wt., whereas changes in body water and protein did not. Thus, nicotine decreases body wt. through its effects on fat stores in the body. After cessation of nicotine, **body fat** rapidly approached levels of control animals.

IT **54-11-5**, Nicotine
RL: BIOL (Biological study)
(body compn. and wt. and food consumption response to)

IT **54-11-5**
RL: BIOL (Biological study)
(tobacco smoke and smoking, nicotine effect on body compn. and wt. and food consumption in relation to)

L4 ANSWER 12 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1990:472897 CAPLUS
DOCUMENT NUMBER: 113:72897
TITLE: Effect of obesity and weight loss on arginine vasopressin response to metoclopramide and nicotine from cigarette smoking
AUTHOR(S): Chiodera, P.; Capretti, L.; Davoli, C.; Caiazza, A.; Bianconi, L.; Coiro, V.
CORPORATE SOURCE: Univ. Parma, Parma, 43100, Italy
SOURCE: Metabolism, Clinical and Experimental (1990), 39(8), 783-6
CODEN: METAAJ; ISSN: 0026-0495
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors previously reported (1987) an impaired arginine vasopressin (AVP) response to insulin-induced hypoglycemia in obese men, suggesting a hypothalamic-posterior pituitary disorder in obesity. In the present study, the AVP response to other releasing stimuli with a central site of action was examd. The AVP response of 10 obese men to metoclopramide (MCP) or nicotine inhaled with cigarette smoking was compared with that obtained in eight sex- and age-matched controls. The AVP increase during nicotine and MCP tests were significantly lower in the obese patients than in the normal controls. Obese men were restudied after substantial wt. loss. The AVP response to nicotine and MCP administration was significantly higher than before **slimming** and did not differ from that obsd. in the normal wt. subjects. These results demonstrate obesity-related alterations in the AVP responsiveness to nicotine inhaled with cigarette smoking and MCP, supporting the hypothesis for a hypothalamic-posterior pituitary disorder in obesity.
IT **54-11-5**, Nicotine 364-62-5, Metoclopramide
RL: BIOL (Biological study)
(arginine vasopressin of blood plasma response to, cigarette smoking in obese men in relation to)

L4 ANSWER 13 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1988:107973 CAPLUS
DOCUMENT NUMBER: 108:107973
TITLE: Lipid effects of smoking
AUTHOR(S): Mjoes, Ole D.
CORPORATE SOURCE: Inst. Med. Biol., Univ. Tromsoe, Tromso, 9000, Norway
SOURCE: American Heart Journal (1988), 115(1, Pt. 2), 272-5
CODEN: AHJOA2; ISSN: 0002-8703
DOCUMENT TYPE: Journal
LANGUAGE: English
AB I.v. nicotine (I) and smoking raise plasma free fatty acid (FFA) levels through enhanced **lipolysis** resulting from sympathoadrenal stimulation. The study reported here investigated FFA-stimulated myocardial O consumption (MvO2) in intact dogs. About half of the nicotine-induced rise in MvO2 resulted from metabolic stimulation by high concns. of FFA, and the remainder was a result of enhanced mech. activity of the heart directly produced by nicotine. In intact dogs, the increase in myocardial O requirement resulting from excess myocardial FFA uptake also increased the severity of myocardial ischemic injury after acute coronary occlusion. Human studies with men who had smoked for >10 yr showed that smokers had lower plasma high-d. lipoprotein cholesterol fractions 2 and 3. High-d.

lipoprotein fraction 2 is reported to be antiatherogenic. Thus, smoking appears to have at least 2 lipid effects that may promote coronary heart disease and atherosclerosis.

IT **54-11-5**, Nicotine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(fatty acids of heart response to, cigarette smoking in relation to)

L4 ANSWER 14 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1984:63007 CAPLUS
DOCUMENT NUMBER: 100:63007
TITLE: Maternal adipose tissue response to nicotine administration in the pregnant rat: effects on fetal **body fat** and cellularity
AUTHOR(S): Williams, Christine M.; Kanagasabai, Tazeen
CORPORATE SOURCE: Dep. Biol. Sci., North East Surrey Coll. Technol., Ewell/Surrey, KT17 3DS, UK
SOURCE: British Journal of Nutrition (1984), 51(1), 7-13
CODEN: BJNUAV; ISSN: 0007-1145
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Maternal adipose tissue response to nicotine administration in the pregnant rat: effects on fetal **body fat** and cellularity
AB The effect of nicotine [**54-11-5**] on fetal growth was studied in rats and related to the actions of the drug on maternal adipose metab. Animals were weighed at regular intervals and killed on day 20 of pregnancy. Rates of maternal adipose tissue **lipolysis** and lipogenesis were measured. Fetal and placental wts. were recorded and anal. of fetal body water, fat, protein, and DNA carried out. Wt. gains of mothers in the nicotine group were less in the 1st and 2nd weeks of pregnancy, but similar to controls in the 3rd week. Fetal body wts., DNA, protein, and percentage water contents were similar in both groups. Mean fetal **body fat** (g/kg) was significantly higher in the nicotine group (96.2) compared with controls (72.0). Rates of maternal **lipolysis** were also higher in the nicotine group. The cause of these differences and their effects on maternal and fetal well-being is discussed.
IT **54-11-5**
RL: BIOL (Biological study)
(embryo growth response to, maternal adipose tissue metab. in relation to)

L4 ANSWER 15 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1975:438571 CAPLUS
DOCUMENT NUMBER: 83:38571
TITLE: Effects of nicotine and inhalation of cigarette smoke on total body oxygen consumption in dogs
AUTHOR(S): Ilebekk, A.; Miller, N. E.; Mjos, O. D.
CORPORATE SOURCE: Ulleval Hosp., Univ. Oslo, Oslo, Norway
SOURCE: Scandinavian Journal of Clinical and Laboratory Investigation (1975), 35(1), 67-72
CODEN: SJCLAY; ISSN: 0036-5513
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects on total body O consumption of nicotine (I) [**54-11-5**] infused intravenously and of simulated cigarette smoking were studied in intact anesthetized dogs. I infusion and simulated cigarette smoking raised total body O consumption by 9% and 6%, resp., and arterial concn. of free fatty acids (FFA) by 29% and 12%. When I infusion and simulated cigarette smoking were repeated during inhibition of **lipolysis** with β-pyridylcarbinol, no rise in total body O consumption occurred,

although the mean aortic blood pressure and heart rate remained elevated to levels similar to those during intact **lipolysis**. Thus, the rise in total body O consumption induced by intravenously infused I or simulated cigarette smoking was probably mediated through increased mobilization and consumption of FFA.

IT **54-11-5**
RL: BIOL (Biological study)
(respiration stimulation by)

L4 ANSWER 16 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1974:446090 CAPLUS
DOCUMENT NUMBER: 81:46090
TITLE: Resistance of rat fetuses to nicotine-induced **lipolysis**
AUTHOR(S): Mosier, H. David., Jr.; Capodanno, Carmen C.; Li, Ivy O. W.; Magruder, Caroline S.; Jansons, Regina A.
CORPORATE SOURCE: Dep. Pediatr., Univ. California, Irvine, CA, USA
SOURCE: Teratology (1974), 9(2), 239-45
CODEN: TJADAB; ISSN: 0040-3709
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Resistance of rat fetuses to nicotine-induced **lipolysis**
AB Dietary or i.p. administration of nicotine (I) [**54-11-5**] to pregnant rats did not affect fetal plasma levels of triglycerides, phospholipids, cholesterol [57-88-5] or free fatty acids. However, injected I did seem to increase epinephrine [51-43-4] release by fetal adrenals. The resistance to change of fetal fatty acids after I is probably dueto resistance to epinephrine-induced elevation of fetal fatty acids. The placenta may be responsible for maintaining the steady state in fetal plasma lipids.
ST embryo **lipolysis** nicotine; epinephrine adrenal embryo nicotine; pregnancy nicotine embryo **lipolysis**
IT Pregnancy
(nicotine-induced **lipolysis** in, embryonic resistance to)
IT Embryo
(nicotine-induced **lipolysis** in, resistance to)
IT **54-11-5**
RL: BIOL (Biological study)
(**lipolysis** from, embryonic resistance to)

L4 ANSWER 17 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1974:486189 CAPLUS
DOCUMENT NUMBER: 81:86189
TITLE: Effect of nicotine on severity of acute myocardial ischemic injury in dogs
AUTHOR(S): Ilebekk, A.; Mjoes, O. D.
CORPORATE SOURCE: Ulleval Hosp., Univ. Oslo, Oslo, Norway
SOURCE: Scandinavian Journal of Clinical and Laboratory Investigation (1974), 33(2), 145-51
CODEN: SJCLAY; ISSN: 0036-5513
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Nicotine (I) [**54-11-5**] (8-10 μg/kg/min, i.v.) during coronary artery occlusion increased the sum of ST-segment elevations (ΣST) from 5-12 sites in anesthetized open-chest dogs. Arterial concn. of free fatty acids rose from 251 +- 31 to 323 +- 50 (μequiv./l). When **lipolysis** was inhibited with β-pyridylcarbinol I failed to raise ΣST at coronary occulsion, nor did the arterial concn. of free fatty acids rise. The increased severity of acute myocardial ischemic injury induced by I is probably related to increased myocardial O requirements caused by excess

myocardial consumption of free fatty acids.
IT **54-11-5**
RL: BIOL (Biological study)
(heart ischemia response to, fatty acid metab. in relation to)

L4 ANSWER 18 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1974:78889 CAPLUS
DOCUMENT NUMBER: 80:78889
TITLE: Effects of nicotine on myocardial metabolism and performance in dogs
AUTHOR(S): Mjos, O. D.; Ilebekk, A.
CORPORATE SOURCE: Inst. Exp. Med. Res., Ulleval Hosp., Oslo, Norway
SOURCE: Scandinavian Journal of Clinical and Laboratory Investigation (1973), 32(1), 75-80
CODEN: SJCLAY; ISSN: 0036-5513
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of an i.v. nicotine (I) [**54-11-5**] infusion on myocardial O consumption was studied before and during inhibition of **lipolysis** with β-pyridylcarbinol. Although mech. responses to I rose similarly in both settings, myocardial O consumption increased by 4.1 before and 2.1 ml/min/100 g during the inhibition of **lipolysis**. Approx. 50% of the I-induced rise in myocardial O consumption was related to enhanced mech. activity of the heart, the remainder being attributable to a metabolic stimulation by high concns. of free fatty acid.
IT **54-11-5**
RL: BIOL (Biological study)
(heart metab. in response to)

L4 ANSWER 19 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1973:11620 CAPLUS
DOCUMENT NUMBER: 78:11620
TITLE: Effect of blocking and stimulation of the nicotinic cholinergic system on the level of free fatty acids, ketone bodies, and sugar in rat serum
AUTHOR(S): Gurin, V. N.; Denisenko, P. P.
CORPORATE SOURCE: Minsk. Med. Inst., Minsk, USSR
SOURCE: Farmakologiya i Toksikologiya (Moscow) (1972), 35(5), 615-19
CODEN: FATOAO; ISSN: 0014-8318
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB Administration to rats of the central N-cholinergic blocking agents pediphen [3146-15-4] or IEM 506 [13426-07-8], or of the peripheral N-cholinergic blocking agent benzohexonium [971-60-8], decreased the blood ketone body and sugar levels; pediphen tended to increase the blood free fatty acid (FFA) content. In immobilized rats, (1) the rise in FFA was lessened by benzohexonium and pediphen; (2) the decrease in ketone bodies was lessened by all 3 agents; and (3) the increase in blood sugar was lessened by benzohexonium. Stimulation by nicotine (I) [**54-11-5**] of central N-cholinergic receptors tended to increase blood FFA and sugar levels and decreased the ketone body level; all 3 effects were lessened by pediphen or benzohexonium. Adrenalectomized rats given I showed a decrease in FFA and sugar levels in blood.
ST cholinergic blocker fatty acid blood; pediphen ketone body blood; IEM 506 sugar blood; benzohexonium **lipolysis**; nicotine **lipolysis**
IT **54-11-5** 971-60-8 3146-15-4 13426-07-8
RL: BIOL (Biological study)
(lipid metabolism regulation by nicotinic cholinergic nervous system response to)

L4 ANSWER 20 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1970:433695 CAPLUS
DOCUMENT NUMBER: 73:33695
TITLE: Changes in serum triglyceride and cholesterol levels independently of free fatty acid after **lipolysis** inhibitors
AUTHOR(S): Tamasi, Gyula; Borsy, Jozsef; Gyenge, R.
CORPORATE SOURCE: Res. Inst. Pharm. Chem., Budapest, Hung.
SOURCE: Biochemical Pharmacology (1970), 19(5), 1826-30
CODEN: BCPCA6; ISSN: 0006-2952
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Changes in serum triglyceride and cholesterol levels independently of free fatty acid after **lipolysis** inhibitors
ST **lipolysis** inhibitors; inhibitors **lipolysis**; triglycerides **lipolysis**; fatty acids **lipolysis**; cholesterol **lipolysis**; serum lipids metab; nicotinic acid serum lipids
IT Blood serum
(fatty acids of, **lipolysis** inhibitor effect on, cholesterol and glycerides in relation to)
IT Glycerides, biological studies
RL: BIOL (Biological study)
(of blood serum, **lipolysis** inhibitor effect on)
IT Fatty acids, biological studies
RL: BIOL (Biological study)
(of blood serum, **lipolysis** inhibitor effect on, cholesterol and glycerides in relation to)
IT **54-11-5**, biological studies 54-86-4 98-96-4 98-97-5 402-61-9 486-74-8 931-03-3
RL: BIOL (Biological study)
(fatty acids of blood serum in response to, cholesterol and glycerides in relation to)
IT 57-88-5, biological studies
RL: BIOL (Biological study)
(of blood serum, **lipolysis** inhibitor effect on)

L4 ANSWER 21 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1971:138257 CAPLUS
DOCUMENT NUMBER: 74:138257
TITLE: Lipid metabolism in atherosclerosis. II. Effect of nicotine on the development of experimental atherosclerosis
AUTHOR(S): Kajiyama, Goro
CORPORATE SOURCE: Sch. Med., Univ. Hiroshima, Hiroshima, Japan
SOURCE: Hiroshima Daigaku Igaku Zasshi (1970), 18(1-2), 21-30
CODEN: HDIZAB; ISSN: 0018-2087
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
ST atherosclerosis lipid metab; nicotine triglyceride aorta; vascular **lipolysis** nicotine
IT **54-11-5**, biological studies
RL: BIOL (Biological study)
(glyceride metabolism by arteries in response to, in atherosclerosis)

L4 ANSWER 22 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1970:98816 CAPLUS
DOCUMENT NUMBER: 72:98816
TITLE: Correlation between the effect of drugs on plasma free

fatty acids and on tissue triglycerides
AUTHOR(S): Bizzi, Adalgisa; Garattini, Silvio
CORPORATE SOURCE: Ist. Ric. Farmacol. "Mario Negri", Milan, Italy
SOURCE: Advan. Exp. Med. Biol. (1969), Volume 4, 201-11. Editor(s): Holmes, William L. Plenum Press: New York, N. Y.
CODEN: AEMBAP
DOCUMENT TYPE: Conference
LANGUAGE: English

AB The findings that free fatty acids (FFA) deriving from adipose tissue may represent an important substrate for the synthesis of liver triglycerides suggests the possibility of controlling triglyceride metabolism by affecting the release of FFA from adipose tissue. An i.v. infusion into mice of fatty acids in a proper medium restores the liver triglycerides deposition in those cases where **lipolysis** has been blocked by 5-carboxy-3-methylpyrazole (I). The results indicated that I and nicotinic acid decreased plasma FFA and heart triglycerides whereas kidney and lung triglycerides were not affected. A rise of plasma FFA obtained by the administration of ACTH to mice increased triglyceride concn. in heart and kidneys. The simultaneous administration of I inhibited the rise of plasma FFA and the triglyceride accumulation. Only the white adipose tissue was important for the availability of FFA necessary for the synthesis of triglycerides. I and nicotinic acid did not lower FFA in brown adipose tissue at doses effective in decreasing FFA in white adipose tissue and in plasma. Several blockers of **lipolysis** such as I, 3-methylpyrazole-5-carboxamide, 5-carboxy-3-methylisoxazole, and pyridyltetrazole become rapidly inactive after repeated administrations. Such resistance apparently is not assocd. with an altered metabolism of the drug. When 3-methylpyrazole 5-carboxamide lost its capacity to depress plasma FFA, it was also ineffective in lowering plasma and liver triglycerides.

IT **54-11-5**, biological studies 402-61-9 4027-56-9 4857-42-5
RL: BIOL (Biological study)
(in triglyceride metabolism, by organs, fatty acid release by adipose tissue in relation to)

L4 ANSWER 23 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1969:66595 CAPLUS
DOCUMENT NUMBER: 70:66595
TITLE: Effect of nicotine on the mobilization of free fatty acids from adipose tissue in vitro
AUTHOR(S): Kershbaum, Alfred; Osada, Hirofumi; Pappajohn, Douglas J.; Bellet, Samuel
CORPORATE SOURCE: Philadelphia Gen. Hosp., Philadelphia, PA, USA
SOURCE: Experientia (1969), 25(2), 128
CODEN: EXPEAM; ISSN: 0014-4754
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nicotine increased the free fatty acid release from rat epididymal fat pads when it was injected i.p. at 0.2 mg./kg. 15 min. before sacrifice but not when it was added in vitro to the incubation medium at 1-8 μg. L-Epinephrine stimulated **lipolysis** both in vivo when injected i.p. at 0.2 mg./kg. and in vitro at 7.28-14.56 μg. Apparently, nicotine has no direct lipolytic action on rat adipose tissue. The mobilization of free fatty acids by nicotine and tobacco smoke may be a result of sympathoadrenal stimulation.

ST tobacco smoke fats rat; nicotine **lipolysis** adipose tissue; **lipolysis** nicotine adipose tissue; adipose tissue **lipolysis** nicotine

IT 51-43-4, biological studies **54-11-5**, biological studies
RL: BIOL (Biological study)
(fatty acid release response to, in adipose tissue)

L4 ANSWER 24 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1967:515551 CAPLUS
DOCUMENT NUMBER: 67:115551
TITLE: Effect of caffeine, nicotine, and ethanol on **lipolysis** in human adipose tissue
AUTHOR(S): Verdy, Maurice
CORPORATE SOURCE: Hop. Hotel-Dieu, Montreal, Can.
SOURCE: Revue Canadienne de Biologie (1967), 26(3), 179-84
CODEN: RCBIAS; ISSN: 0035-0915
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Effect of caffeine, nicotine, and ethanol on **lipolysis** in human adipose tissue
AB The **lipolysis** rate in human omental adipose tissue was measured in vitro. EtOH had no const. effect, even at 4 mg./ml. Small amts. (10-4M) of caffeine failed to effect **lipolysis** or potentiate the lipolytic effect of adrenaline, but higher doses (10-3M) did exert a strong lipolytic effect. Nicotine at 10-4M had no direct effect, but reduced the lipolytic activity of adrenaline by 20%. 29 references.
ST ADIPOSE TISSUE **LIPOLYSIS** ETHANOL; CAFFEINE **LIPOLYSIS** ADIPOSE; **LIPOLYSIS** ADIPOSE TISSUE ETHANOL; ETHANOL ADIPOSE TISSUE **LIPOLYSIS**; NICOTINE **LIPOLYSIS** ADIPOSE; ADRENALINE **LIPOLYSIS** NICOTINE
IT Adipose tissue, metabolism
(**lipolysis** in, effect of caffeine, ethyl alc. and nicotine on adrenaline-induced)
IT 64-17-5, biological studies
RL: BIOL (Biological study)
(**lipolysis** in adipose tissue and)
IT **54-11-5**, biological studies 58-08-2, biological studies
RL: BIOL (Biological study)
(**lipolysis** response to, in adipose tissue)
IT 51-43-4, biological studies
RL: BIOL (Biological study)
(**lipolysis** response to, in adipose tissue, effect of caffeine, ethyl alc. and nicotine on)

=> s nicotine and (slimming or cellulite or anticellulite or lipolysis)
L5 57 NICOTINE AND (SLIMMING OR CELLULITE OR ANTICELLULITE OR LIPOLYSIS)

=> s l5 not l4
L6 40 L5 NOT L4

=> dup rem l6
DUPLICATE IS NOT AVAILABLE IN 'KOSMET'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L6
L7 24 DUP REM L6 (16 DUPLICATES REMOVED)

=> d l7 ibib kwic 1-24

L7 ANSWER 1 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
Full Text
ACCESSION NUMBER: 2001:908694 CAPLUS
DOCUMENT NUMBER: 136:129315
TITLE: Inhibition of the rise in FFA by Acipimox partially prevents GH-induced insulin resistance in GH-deficient adults
AUTHOR(S): Segerlantz, Mikael; Bramnert, Margareta; Manhem, Per;

Laurila, Esa; Groop, Leif C.
CORPORATE SOURCE: Department of Endocrinology, University Hospital MAS, Malmo, S-205 02, Swed.
SOURCE: Journal of Clinical Endocrinology and Metabolism (2001), 86(12), 5813-5818
CODEN: JCEMAZ; ISSN: 0021-972X
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB To test the hypothesis that GH-induced insulin resistance is mediated by an increase in FFA levels the authors assessed insulin sensitivity after inhibiting the increase in FFA by a **nicotine** acid deriv., Acipimox, in nine GH-deficient adults receiving GH replacement therapy. The patients received in a double blind fashion either Acipimox (500 mg) or placebo before a 2-h euglycemic (plasma glucose, 5.5±0.2 mmol/L) hyperinsulinemic (serum insulin, 28.7±6.3 mU/L) clamp in combination with indirect calorimetry and infusion of [3-3H]glucose. Acipimox decreased fasting FFA by 88% (P = 0.012) and basal lipid oxidn. by 39% (P = 0.015) compared with placebo. In addn., the insulin-stimulated lipid oxidn. was 31% (P = 0.0077) lower during Acipimox than during placebo. Acipimox increased insulin-stimulated total glucose uptake by 36% (P = 0.021) compared with placebo, which mainly was due to a 47% (P = 0.015) increase in glucose oxidn. GH induced insulin resistance is partially prevented by inhibition of **lipolysis** by Acipimox.

IT Lipids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**lipolysis**; inhibition of rise in FFA by Acipimox partially prevents GH-induced insulin resistance in GH-deficient adults)

L7 ANSWER 2 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 2
ACCESSION NUMBER: 2001:408682 BIOSIS
DOCUMENT NUMBER: PREV200100408682
TITLE: Systemic **nicotine** stimulates human adipose tissue **lipolysis** through local cholinergic and catecholaminergic receptors.
AUTHOR(S): Andersson, K.; Arner, P. [Reprint author]
CORPORATE SOURCE: Center for Metabolism and Endocrinology, Huddinge University Hospital, S-141 86, Stockholm, Sweden
Peter.Arner@medhs.ki.se
SOURCE: International Journal of Obesity, (August, 2001) Vol. 25, No. 8, pp. 1225-1232. print.
CODEN: IJOBDP. ISSN: 0307-0565.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Aug 2001
Last Updated on STN: 22 Feb 2002

TI Systemic **nicotine** stimulates human adipose tissue **lipolysis** through local cholinergic and catecholaminergic receptors.

AB OBJECTIVE: To evaluate whether the lipolytic effects of systemic **nicotine** are not only attributed to indirect adrenergic mechanisms, but also to a direct action of **nicotine** on fat cells. DESIGN: The effect of a systemic **nicotine** infusion (0.5 mug/kg/min for 30 min) on **lipolysis** in subcutaneous adipose tissue was investigated in situ in 11 non-obese, non-smoking, healthy male subjects under placebo-controlled conditions. MEASUREMENTS: By using microdialysis probes the glycerol levels (**lipolysis** index) and blood flow were monitored locally in subcutaneous adipose tissue. RESULTS: Plasma **nicotine** levels peaked (7.2 ng/ml) at the end of the infusion. **Nicotine** induced a mean (+-s.e.) percentage

peak increase in adrenaline and noradrenaline plasma levels of 213+-30% (P<0.01) and 118+-5% (P<0.05), respectively. **Nicotine** increased venous plasma glycerol levels by 144+-9% (P<0.001), arterialized plasma glycerol levels by 148+-12% (P<0.001) and adipose glycerol levels by. . . levels by apprx60% (P=0.02). Infusion of saline (ie placebo) had no effect on the parameters mentioned above. CONCLUSION: Systemically administered **nicotine** induces **lipolysis**, in part by activating the classical adrenergic mechanism (mediated by a **nicotine**-induced release of catecholamines stimulating beta-adrenoceptors), and in part by directly activating a nicotinic cholinergic lipolytic receptor located in adipose tissue.

IT . . .
Systems of Organisms
plasma: blood and lymphatics; subcutaneous adipose tissue
IT Chemicals Biochemicals
adrenaline; beta-adrenoceptors; catecholaminergic receptors; glycerol; **nicotine**: lipolytic effect, systemic administration; nicotinic cholinergic lipolytic receptor; noradrenaline
IT Miscellaneous Descriptors
lipolysis
RN 51-43-4 (adrenaline)
56-81-5 (glycerol)
54-11-5 (**nicotine**)
51-41-2 (noradrenaline)

L7 ANSWER 3 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 3
ACCESSION NUMBER: 1997:312218 BIOSIS
DOCUMENT NUMBER: PREV199799620021
TITLE: Pharmacodynamics of acute tolerance to multiple nicotinic effects in humans.
AUTHOR(S): Fattinger, Karin; Verotta, Davide; Benowitz, Neal L. [Reprint author]
CORPORATE SOURCE: Division Clinical Pharmacol. Experimental Therapeutics, Univ. California San Francisco, Box 1220, San Francisco, CA 94143-1220, USA
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (1997) Vol. 281, No. 3, pp. 1238-1246.
CODEN: JPETAB. ISSN: 0022-3565.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 26 Jul 1997
Last Updated on STN: 26 Jul 1997

AB. . . of addiction as well as therapeutic and/or toxic effects of drugs. The development of acute tolerance to various effects of **nicotine** was studied in nine healthy smokers who were abstaining from tobacco. **Nicotine** was infused rapidly to reach a concentration of about 25 ng/ml, followed by a computer-controlled infusion to maintain that concentration. A novel semiparametric model of **nicotine** effects and tolerance was developed. Tolerance to various effects of **nicotine** (increases in heart rate, blood pressure, plasma epinephrine and energy expenditure) occurred within the range of **nicotine** levels found in smokers. However, the rate of tolerance development varied considerably. The half-lives of tolerance ranged from 3.5 min. . . min for systolic blood pressure. There was no apparent tolerance to the effects on free fatty acid concentrations, which reflects **lipolysis**. Differences in the pharmacodynamics of tolerance may reflect differences in rate of desensitization of various subtypes of nicotinic receptors and/or. . .

IT . . .
System (Transport and Circulation); Endocrine System (Chemical Coordination and Homeostasis); Metabolism; Nervous System (Neural

Coordination); Toxicology
IT Chemicals Biochemicals
NICOTINE; EPINEPHRINE
IT Miscellaneous Descriptors
ADDICTION; BLOOD PRESSURE; ENERGY EXPENDITURE; EPINEPHRINE; FREE FATTY ACIDS; HEART RATE; **NICOTINE**; PHARMACODYNAMICS; TOLERANCE; TOXICOLOGY
RN 54-11-5 (**NICOTINE**)
51-43-4 (EPINEPHRINE)

L7 ANSWER 4 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 4
ACCESSION NUMBER: 1997:343497 BIOSIS
DOCUMENT NUMBER: PREV199799642700
TITLE: No acute effects of smoking and **nicotine** nasal spray on **lipolysis** measured by subcutaneous microdialysis.
AUTHOR(S): Eliasson, B. [Reprint author]; Smith, U.; Lonnroth, P.
CORPORATE SOURCE: Lundberg Lab. Diabetes Res., Sahlgrenska Univ. Hosp., S-413 45 Goteborg, Sweden
SOURCE: European Journal of Clinical Investigation, (1997) Vol. 27, No. 6, pp. 503-509.
CODEN: EJCIB8. ISSN: 0014-2972.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Aug 1997
Last Updated on STN: 11 Aug 1997
TI No acute effects of smoking and **nicotine** nasal spray on **lipolysis** measured by subcutaneous microdialysis.
AB Smoking is associated with insulin resistance, dyslipidaemia and markers of the insulin resistance syndrome. This study investigated adipose tissue **lipolysis** in situ by subcutaneous microdialysis twice in 10 healthy, male smokers after smoking four cigarettes over 2h and after the administration of an equal amount of levels, in situ **lipolysis** and adipose tissue blood flow were and lactate levels were significantly higher after smoking, whereas FFA levels were higher after. . . administration of a normal dose of NNS caused only minor metabolic changes. Thus, it does not seem likely that increased **lipolysis** is an important contributor to the dyslipidaemia seen in smokers.
IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Metabolism; Methods and Techniques; Toxicology
IT Chemicals Biochemicals
NICOTINE; INSULIN; GLYCEROL
IT Miscellaneous Descriptors
ANALYTICAL METHOD; CLINICAL ENDOCRINOLOGY; DYSLIPIDEMIA; FREE FATTY ACID; GLYCEROL; INSULIN RESISTANCE SYNDROME; **LIPOLYSIS**; METABOLIC DISEASE; METHODOLOGY; NASAL SPRAY; **NICOTINE**; SMOKING; SUBCUTANEOUS MICRODIALYSIS
RN 54-11-5 (**NICOTINE**)
9004-10-8 (INSULIN)
56-81-5 (GLYCEROL)

L7 ANSWER 5 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 5
ACCESSION NUMBER: 1996:232640 BIOSIS
DOCUMENT NUMBER: PREV199698796769
TITLE: Alterations of **lipolysis** and lipoprotein lipase in chronically **nicotine**-treated rats.
AUTHOR(S): Sztalryd, Carole; Hamilton, Jock; Horwitz, Barbara A.; Johnson, Patricia; Kraemer, Frederic B.
CORPORATE SOURCE: Div. Endocrinol., S-005, Stanford Univ. Med. Cent.,

Stanford, CA 94305-5103, USA
SOURCE: American Journal of Physiology, (1996) Vol. 270, No. 2 PART 1, pp. E215-E223.
CODEN: AJPHAP. ISSN: 0002-9513.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 28 May 1996
Last Updated on STN: 28 May 1996

TI Alterations of **lipolysis** and lipoprotein lipase in chronically **nicotine**-treated rats.

AB These studies examined the cellular mechanisms for lower adiposity seen with **nicotine** ingestion. Rats were infused with **nicotine** or saline for 1 wk and adipocytes isolated from epididymal fat pads. **Nicotine**-infused rats gained 37% less weight and had 21% smaller fat pads. Basal **lipolysis** was 78% higher, whereas the maximal lipolytic response to isoproterenol was blunted in adipocytes from **nicotine**-infused rats. The antilipolytic actions of adenosine and the levels of serum catecholamines were unaffected by **nicotine**. The **nicotine**-induced alteration in **lipolysis** was not associated with any changes in hormone-sensitive lipase. **Nicotine** caused a 30% decrease in lipoprotein lipase (LPL) activity, without any changes in LPL mass or mRNA levels, in epididymal fat in the fed state. In contrast, LPL activity, mass, and mRNA levels in heart were increased by **nicotine** whether animals were fed or fasted. These studies provide evidence for multiple mechanistic events underlying **nicotine**-induced alterations in weight and suggest that **nicotine** diverts fat storage away from adipose tissue and toward utilization by muscle.

IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Metabolism; Pharmacology; Skeletal System (Movement and Support)

IT Chemicals Biochemicals
LIPOPROTEIN LIPASE; **NICOTINE**

IT Miscellaneous Descriptors
ADIPOSE TISSUE; **NICOTINE**; WEIGHT GAIN

RN 9004-02-8 (LIPOPROTEIN LIPASE)
54-11-5 (**NICOTINE**)

L7 ANSWER 6 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 6
ACCESSION NUMBER: 1995:432568 BIOSIS
DOCUMENT NUMBER: PREV199598446868
TITLE: Cholinoceptor-mediated effects on glycerol output from human adipose tissue using in situ microdialysis.
AUTHOR(S): Andersson, Kurt; Arner, Peter [Reprint author]
CORPORATE SOURCE: Dep. Med., Karolinska Inst., S-14186 Huddinge, Sweden
SOURCE: British Journal of Pharmacology, (1995) Vol. 115, No. 7, pp. 1155-1162.
CODEN: BJPCBM. ISSN: 0007-1188.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Oct 1995
Last Updated on STN: 10 Oct 1995

AB 1. Possible cholinoceptor-mediated effects on **lipolysis** were investigated in vivo in human subcutaneous adipose tissue of non-obese, non-smoking, healthy subjects, by use of microdialysis. Cholinomimetic and sympathomimetic agents were added to the ingoing dialysate solvent. 2. Addition of **nicotine** to the perfusion solvent caused a concentration-dependent reversible increase in the levels of glycerol in the dialysate (**lipolysis** index). The opposite effect (also concentration-dependent and reversible) was caused by the addition of carbachol. The maximum effects were 100% stimulation and 50% inhibition,

respectively, by **nicotine** and carbachol. Neither **nicotine** nor carbachol stimulated nutritive blood flow in adipose tissue (as measured with an ethanol escape technique). 3. The **nicotine** effect in situ was concentration-dependently counteracted by the nicotinic cholinoceptor antagonist, mecamylamine. Likewise, the carbachol effect was concentration-dependently counteracted by. . . was pretreated with phentolamine plus propranolol in order to obtain a complete alpha and beta-adrenoceptor blockade, the subsequent addition of **nicotine** or carbachol still induced an increase and decrease in dialysate glycerol levels (lipolytic or antilipolytic effects), respectively. When adipose tissue. . . or atropine, the subsequent addition of acetylcholine caused a reversible decrease and increase, respectively, of the dialysate glycerol levels. 5. **Nicotine** and carbachol had no effects on glycerol release from human isolated subcutaneous fat cells that were incubated in vivo. 6.. . .

IT . . .
Coordination and Homeostasis); Metabolism; Nervous System (Neural Coordination); Nutrition; Skeletal System (Movement and Support)

IT Chemicals Biochemicals
GLYCEROL; CARBACHOL; **NICOTINE**; MECAMYLAMINE; ATROPINE; PHENTOLAMINE; PROPRANOLOL; ACETYLCHOLINE

IT Miscellaneous Descriptors
ACETYLCHOLINE; ATROPINE; CARBACHOL; FAT CELL; **LIPOLYSIS**; MECAMYLAMINE; **NICOTINE**; NUTRITIVE BLOOD FLOW; PHENTOLAMINE; PROPRANOLOL

RN 56-81-5 (GLYCEROL)
51-83-2 (CARBACHOL)
54-11-5 (**NICOTINE**)
60-40-2 (MECAMYLAMINE)
51-55-8 (ATROPINE)
50-60-2 (PHENTOLAMINE)
525-66-6 (PROPRANOLOL)
51-84-3 (ACETYLCHOLINE)

L7 ANSWER 7 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 7
Full Text

ACCESSION NUMBER: 1995:296452 CAPLUS
DOCUMENT NUMBER: 122:99192
TITLE: Metabolic interactions between surplus dietary energy intake and cigarette smoking or its cessation
AUTHOR(S): Neese, Richard A.; Benowitz, Neal L.; Hoh, Rebecca; Faix, Dennis; LaBua, Alice; Pun, Keynes; Hellerstein, Marc K.
CORPORATE SOURCE: Dep. Med., Univ. California, San Francisco, CA, 94110, USA
SOURCE: American Journal of Physiology (1994), 267(6, Pt. 1), E1023-E1034
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cigarette smoking (CS) alters lipid metab. and is assocd. clin. with an atherogenic lipid profile. It has been shown that, under controlled eucaloric dietary conditions, CS stimulates **lipolysis** without increasing oxidn. of fat and that cessation of CS does not result in a rebound tendency to synthesize or store fat. It was asked here whether the ad libitum intake of surplus dietary energy interacts with the metabolic effects of CS or its cessation. Eight male heavy smokers were allowed ad libitum food intake in a metabolic ward, 1 wk in CS phase and 1 wk in non-CS phase, followed by 4 wk of outpatient non-CS and a repeat 7-day study. De novo hepatic lipogenesis (DNL), **lipolysis**, substrate cycling of free fatty acids (FFA), hepatic glucose prodn., and energy expenditure were measured by using a multiple stable-isotope infusion protocol and

indirect calorimetry. Surplus dietary energy intake (>150% of predicted energy needs) occurred in five of eight subjects (2 subjects >5500 kcal/day, 3 subjects >4000 kcal/day) with wt. gain of 1-4 kg/wk, but with no difference between CS and non-CS phases. Acute CS significantly increased (P <0.05) serum FFA concns. (58%), FFA flux (63%), and glycerol flux (36%); nonsignificantly increased extra-adipocyte (hepatic) esterification of FFA (125%, P = 0.10) and resting energy expenditure (4.1%, P = 0.22); and did not change adipocyte reesterification of FFA or whole-body oxidn. of fat. Basal metabolic parameters (after overnight abstention from CS) did not differ between phases. Fractional DNL correlated significantly with excess energy intake (r2 = 0.39) and with percentage of total energy needs provided by carbohydrate (r2 = 0.47). The absence or presence of CS did not affect the increase in fractional DNL in subjects with excess energy intake, however. Thus, cessation of CS does not result in a rebound tendency to synthesis or storage of fat, even in the presence of pos. short-term energy balance, contrary to previous suggestions. Moreover, stimulation of **lipolysis** by CS does not increase oxidn. of fat and thereby protect against fat deposition under conditions of surplus energy intake. The prevention of wt. gain after cessation of CS, whether or not **nicotine** is provided, should focus on energy balance (calorigenesis as well as intake) rather than specific alterations in lipid metab.

L7 ANSWER 8 OF 24 MEDLINE on STN
Full Text

ACCESSION NUMBER:	94163200 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 8118470
TITLE:	Changes in circulating lipid and carbohydrate metabolites following systemic **nicotine** treatment in healthy men.
AUTHOR:	Andersson K; Eneroth P; Arner P
CORPORATE SOURCE:	Department of Medicine, Karolinska Institute, Huddinge Hospital, Stockholm, Sweden.
SOURCE:	International journal of obesity and related metabolic disorders : journal of the International Association for the Study of Obesity, (1993 Dec) 17 (12) 675-80. Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY:	ENGLAND: United Kingdom
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	199404
ENTRY DATE:	Entered STN: 19940412 Last Updated on STN: 19940412 Entered Medline: 19940406

TI Changes in circulating lipid and carbohydrate metabolites following systemic **nicotine** treatment in healthy men.

AB In the present study the influence of low doses of intravenous **nicotine** administration on hormonal and metabolic events was studied in man in view of the clinical implications of moderate smoking on the development of hyperlipidemia. Hormonal, metabolic and cardiovascular effects of a 30 min intravenous **nicotine** infusion (0.25 or 0.5 microgram/kg/min) were determined in seven non-smoking, healthy, normal weight male individuals after an overnight fast. **Nicotine** caused a significant dose-dependent increase in the plasma levels of **nicotine**, cotinine, noradrenaline, adrenaline, glycerol and free fatty acids (FFA). The serum **nicotine** concentrations peaked at the end of the infusion followed by a gradual decline, although they were still increased 90 min after cessation of infusion. Serum cotinine levels (the main **nicotine** metabolite) continuously increased during the experiment and statistically significant increases were found from 30 min after the start of infusion of **nicotine**. Serum noradrenaline, adrenaline, glycerol and FFA levels had increased significantly by 15 min of **nicotine** infusion. **Nicotine**

produced significant elevations of adrenaline, glycerol and FFA concentrations at both doses (maximal increments of 247, 184 and 153%, respectively) and the peak effect occurred at 30 min. However, noradrenaline levels only responded to the high **nicotine** dose and the maximal increment (168%) was already found at 15 min. The increments of noradrenaline and adrenaline failed to elicit changes in systolic and diastolic blood pressure or heart rate. **Nicotine** did not alter plasma levels of glucagon, insulin, glucose, pyruvate or lactate and a non-significant increase in serum cortisol and. . .

CT . . .
Catecholamines: BL, blood
Hemodynamic Processes: DE, drug effects
Hormones: BL, blood
Hyperlipidemia: CI, chemically induced
Infusions, Intravenous
*Lipids: BL, blood
***Lipolysis: DE, drug effects**
Nicotine: AD, administration dosage
Nicotine: BL, blood
***Nicotine: PD, pharmacology**
RN **54-11-5 (Nicotine)**

L7 ANSWER 9 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 8
ACCESSION NUMBER: 1990:433091 BIOSIS
DOCUMENT NUMBER: PREV199090093892; BA90:93892
TITLE: EFFECT OF OBESITY AND WEIGHT LOSS ON ARGININE VASOPRESSIN RESPONSE TO METOCLOPRAMIDE AND **NICOTINE** FROM CIGARETTE SMOKING.
AUTHOR(S): CHIODERA P [Reprint author]; CAPRETTI L; DAVOLI C; CAIAZZA A; BIANCONI L; COIRO V
CORPORATE SOURCE: CATTEDRA DI ENDOCRINOL E PATOL COSTITUZIONALE, UNIV DI PARMA, VIA GRAMSCI 14, 43100 PARMA, ITALY
SOURCE: Metabolism Clinical and Experimental, (1990) Vol. 39, No. 8, pp. 783-786.
CODEN: METAAJ. ISSN: 0026-0495.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 22 Sep 1990
Last Updated on STN: 23 Sep 1990
TI EFFECT OF OBESITY AND WEIGHT LOSS ON ARGININE VASOPRESSIN RESPONSE TO METOCLOPRAMIDE AND **NICOTINE** FROM CIGARETTE SMOKING.
AB. . . other releasing stimuli with a central site of action. The AVP response of 10 obese men to metoclopramide (MCP) or **nicotine** inhaled with cigarette smoking was compared with that obtained in eight sex- and age-matched controls. The AVP increase during **nicotine** and MCP tests were significantly lower in the obese patients than in the normal controls. Obese men were restudied after substantial weight loss. The AVP response to **nicotine** and MCP administration was significantly higher than before **slimming** and did not differ from that observed in the normal weight subjects. These results demonstrate obesity-related alterations in the AVP responsiveness to **nicotine** inhaled with cigarette smoking and MCP, supporting the hypothesis for a hypothalamic-posterior pituitary disorder in obesity.
RN 113-79-1 (ARGININE VASOPRESSIN)
364-62-5 (METOCLOPRAMIDE)
54-11-5 (**NICOTINE**)
9004-10-8 (INSULIN)

L7 ANSWER 10 OF 24 MEDLINE on STN

Full Text
ACCESSION NUMBER: 88103268 MEDLINE
DOCUMENT NUMBER: 88103268 PubMed ID: 3336994
TITLE: Lipid effects of smoking.
AUTHOR: Mjos O D
CORPORATE SOURCE: Department of Physiology, University of Tromso, Norway.
SOURCE: AMERICAN HEART JOURNAL, (1988 Jan) 115 (1 Pt 2) 272-5.
Journal code: 0370465. ISSN: 0002-8703.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198802
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19900305
Entered Medline: 19880205

AB Cigarette smoking is believed to cause harmful cardiovascular and atherogenic effects resulting from changes in lipid metabolism. Intravenous **nicotine** and smoking raise plasma free fatty acid (FFA) levels through enhanced **lipolysis** resulting from sympathoadrenal stimulation. The study reported here investigated FFA-stimulated myocardial oxygen consumption (MVO2) in intact dogs. It was found that about half of the **nicotine**-induced rise in MVO2 resulted from metabolic stimulation by high concentrations of FFA, and the remainder was a result of enhanced mechanical activity of the heart directly produced by **nicotine**. In intact dogs, the increase in myocardial oxygen requirement resulting from excess myocardial FFA uptake also increased the severity of. . .

CT Check Tags: Animal; Human
Adult
*Cholesterol: BL, blood
*Coronary Disease: ET, etiology
Dogs
Hemodynamics: DE, drug effects
***Lipolysis: DE, drug effects**
Middle Age
***Nicotine: PO, poisoning**
*Smoking
*Triglycerides: BL, blood

RN **54-11-5 (Nicotine)**; 57-88-5 (Cholesterol)

L7 ANSWER 11 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 9
ACCESSION NUMBER: 1984:265161 BIOSIS
DOCUMENT NUMBER: PREV198478001641; BA78:1641
TITLE: MATERNAL ADIPOSE TISSUE RESPONSE TO **NICOTINE** ADMINISTRATION IN THE PREGNANT RAT EFFECTS ON FETAL BODY FAT AND CELLULARITY.
AUTHOR(S): WILLIAMS C M [Reprint author]; KANAGASABAI T
CORPORATE SOURCE: DEP BIOLOGICAL SCI, NORTH EAST SURREY COLLEGE TECHNOLOGY, REIGATE RD, EWELL, SURREY KT17 3DS, UK
SOURCE: British Journal of Nutrition, (1984) Vol. 51, No. 1, pp. 7-14.
CODEN: BJNUAV. ISSN: 0007-1145.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

TI MATERNAL ADIPOSE TISSUE RESPONSE TO **NICOTINE** ADMINISTRATION IN THE PREGNANT RAT EFFECTS ON FETAL BODY FAT AND CELLULARITY.

AB **Nicotine** may be a causative factor in the intrauterine growth retardation associated with smoking in pregnancy. A study was set up to

ascertain the effect of **nicotine** on fetal growth and whether this could be related to the actions of this drug on maternal adipose tissue metabolism. Sprague-Dawley rats were mated and assigned to control and **nicotine** groups, the latter receiving **nicotine** in the drinking-water throughout pregnancy. Animals were weighed at regular intervals and killed on day 20 of pregnancy. Rates of maternal adipose tissue **lipolysis** and lipogenesis were measured. Fetal and placental weights were recorded and analysis of fetal body water, fat, protein and DNA carried out. Weight gains of mothers in the **nicotine** group were less in the 1st and 2nd wk of pregnancy, but similar to controls in the 3rd wk. Fetal. . . protein and percentage water contents were similar in both groups. Mean fetal body fat (g/kg) was significantly higher in the **nicotine** group (96.2 ± 5.1) compared with controls (72.0 ± 2.9). Rates of maternal **lipolysis** were also higher in the **nicotine** group. The cause of these differences and their effects on maternal and fetal well-being is discussed.

RN 54-11-5 (**NICOTINE**)

L7 ANSWER 12 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text

DUPLICATE 10

ACCESSION NUMBER: 1984:74342 BIOSIS
DOCUMENT NUMBER: PREV198426074342; BR26:74342
TITLE: DOES NALOXONE ALWAYS ACT AS AN OPIATE ANTAGONIST.
AUTHOR(S): BADAWY A A-B [Reprint author]; EVANS M; PUNJANI N F; MORGAN C J
CORPORATE SOURCE: SOUTH GLAMORGAN HEALTH AUTHORITY, ADDICTION UNIT RES LAB, WHITCHURCH HOSP, CARDIFF CF4 7XB, WALES, UK
SOURCE: Life Sciences, (1983) Vol. 33, No. SUPPL. 1, pp. 739-742. Meeting Info.: INTERNATIONAL NARCOTIC RESEARCH CONFERENCE, GARMISCH-PARTENKIRCHEN, JUNE 26-JULY 1, 1983. LIFE SCI. CODEN: LIFSAK. ISSN: 0024-3205.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
IT . . .
RAT LIVER BRAIN ENDOGENOUS OPIATES RECEPTORS NADPH NAD TRYPTOPHAN PYRROLASE 5 AMINO LEVULINATE SYNTHASE ACTIVITY METABOLIC-DRUG MORPHINE CENTRAL DEPRESSANT ETHANOL **NICOTINE** PHENO BARBITONE DEPENDENCE **LIPOLYSIS** PHARMACODYNAMICS
RN 465-65-6 (NALOXONE)
53-57-6 (NADPH)
53-84-9 (NAD)
9014-51-1 (TRYPTOPHAN PYRROLASE)
9037-14-3 (5-AMINOLEVULINATE SYNTHASE)
57-27-2 (MORPHINE)
64-17-5 (ETHANOL)
54-11-5 (**NICOTINE**)
50-06-6 (PHENOBARBITONE)

L7 ANSWER 13 OF 24 MEDLINE on STN
Full Text

ACCESSION NUMBER: 82072469 MEDLINE
DOCUMENT NUMBER: 82072469 PubMed ID: 7308122
TITLE: [Metabolic changes in the liver as affected by nicotinic acid].
Metabolitni promeni v cherniia drob pod vliianie na nikotinovata kiselina.
AUTHOR: Orbetsova V
SOURCE: EKSPERIMENTALNA MEDITSINA I MORFOLOGIIA, (1981) 20 (3) 143-50.
Journal code: 0007506. ISSN: 0367-0643.

PUB. COUNTRY: Bulgaria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Bulgarian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198202
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19820212

AB . . . 2mM/kg of body weight. She examined at the 1th, 3th, 6th and 24th hour the changes in the levels of **nicotine**-amide coenzymes (NAD, NAD-H and NADP), adenine nucleotides (ATP, ADP and AMP), the metabolic lactate and pyruvate and the enzymes LDH,. . . (NA) complex metabolic changes occurred in liver, due to its basic effects-stimulation of biosynthesis of nicotinamide coenzymes and inhibition of **lipolysis** in the fatty tissue. Most probably the effect on the biosynthesis of NAD was primary, which showed later substantial regulatory influence both on **lipolysis** in the fatty acid and on the metabolization of mobilizing lipids on behalf of the liver. Parallel occurring metabolic processes. . .

L7 ANSWER 14 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 11
Full Text
ACCESSION NUMBER: 1978:540376 CAPLUS
DOCUMENT NUMBER: 89:140376
TITLE: Pharmacological profile of BR-931, a new hypolipidemic agent that increases high-density lipoproteins
AUTHOR(S): Sirtori, Cesare R.; Gomarasca, Piero; D'Atri, Gaetano; Cerutti, Silvano; Tronconi, Gianni; Scolastico, Carlo
CORPORATE SOURCE: Cent. E. Grossi Paoletti, Univ. Milan, Milan, Italy
SOURCE: Atherosclerosis (Shannon, Ireland) (1978), 30(1), 45-56
CODEN: ATHSBL; ISSN: 0021-9150
DOCUMENT TYPE: Journal
LANGUAGE: English

AB BR-931 [4-chloro-6-(2,3-xylidino)-2-pyrimidinylthio-N-(β-hydroxyethyl)acetamide](I) [65089-17-0], a new hypolipidemic agent of low toxicity, was evaluated in several tests of **lipolysis** and hyperlipidemia in rats, and in the cholesterol-induced atherosclerosis in rabbits. Significant hypolipidemic activity was obsd. in rats with doses of 12.5 to 50 mg I/kg. In the Triton-induced hyperlipidemia, 50 mg I/kg was equieffective as 200 mg of clofibrate (CPIB)/kg. In contrast with CPIB, I exerted a powerful antilipolytic activity against epinephrine, ACTH, **nicotine**, and cold exposure. I was particularly effective in diet-induced hyperlipidemias. EtOH lipemia was totally prevented by the agent at 100 mg/kg. With Nath's diet, doses as low as 25 mg/kg decreased hypercholesterolemia and hypertriglyceridemia. In these last 2 tests, the distribution of lipoprotein cholesterol was not detd. CPIB did not affect high-density lipoprotein (HDL) cholesterol levels thathad been decreased by the diets; in contrast, I, at doses of 50 mg/kg, brought the HDL/total cholesterol ratio back toward normal. A significant HDL cholesterol increase, together with some decrease of atheromatosis, was also obsd. in cholesterol-fed rabbits. I, a potent inducer of liver peroxisomes and of mitochondrial carnitine acetyltransferase, appears to be a hypolipidemic agent of high efficacy and low toxicity for the clin. treatment of hyperlipidemias and atherosclerosis.

L7 ANSWER 15 OF 24 MEDLINE on STN
Full Text
ACCESSION NUMBER: 76079987 MEDLINE
DOCUMENT NUMBER: 76079987 PubMed ID: 1197881
TITLE: Changes in fatty acid composition of cardiac lipids accompanying myocardial necrosis.

AUTHOR: Gudbjarnason S; Oskarsdottir G
SOURCE: RECENT ADVANCES IN STUDIES ON CARDIAC STRUCTURE AND METABOLISM, (1975) 6 193-203.
Journal code: 0325677. ISSN: 0363-5872.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197602
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19970203
Entered Medline: 19760227

AB . . . an important role in myocardial cell damage. Myocardial damage following administration of isoproterenol is increased when there is enhanced intramyocardial **lipolysis** and an increased amount of polyunsaturated fatty acids in membrane phospholipids. Mocardial damage is decreased when there is reduced intramyocardial **lipolysis** and the membrane response makes the membranes less permeable and more stable.

CT . . .
Isoproterenol
*Lipids: ME, metabolism
Myocardial Diseases: CI, chemically induced
*Myocardial Diseases: ME, metabolism
*Myocardium: ME, metabolism
Myocardium: PA, pathology
Necrosis
Nicotine: PD, pharmacology
Phospholipids: ME, metabolism
Rats
Triglycerides: ME, metabolism

RN **54-11-5 (Nicotine)**; 7683-59-2 (Isoproterenol); 8001-69-2 (Cod Liver Oil)

L7 ANSWER 16 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 12
ACCESSION NUMBER: 1975:198965 BIOSIS
DOCUMENT NUMBER: PREV197560028961; BA60:28961
TITLE: EFFECTS OF **NICOTINE** AND INHALATION OF CIGARETTE SMOKE ON TOTAL BODY OXYGEN CONSUMPTION IN DOGS.
AUTHOR(S): ILEBEKK A; MILLER N E; MJOS O D
SOURCE: Scandinavian Journal of Clinical and Laboratory Investigation, (1975) Vol. 35, No. 1, pp. 67-72.
CODEN: SJCLAY. ISSN: 0036-5513.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

TI EFFECTS OF **NICOTINE** AND INHALATION OF CIGARETTE SMOKE ON TOTAL BODY OXYGEN CONSUMPTION IN DOGS.
IT Miscellaneous Descriptors
BETA PYRIDYL CARBINOL METAB-DRUG **LIPOLYSIS** BLOOD PRESSURE HEART RATE FREE FATTY-ACIDS
RN 54-11-5 (**NICOTINE**)
7782-44-7 (OXYGEN)
100-55-0 (BETA PYRIDYL CARBINOL)

L7 ANSWER 17 OF 24 MEDLINE on STN
Full Text
ACCESSION NUMBER: 74296172 MEDLINE
DOCUMENT NUMBER: 74296172 PubMed ID: 4849795
TITLE: [Studies on the mechanism of **nicotine**-induced **lipolysis** in man].
Untersuchungen zum Mechanismus der durch Nikotin

induziertem Lipolyse beim Menschen.
AUTHOR: Ratzmann K P; Meyer L W; Riemer D
SOURCE: ZEITSCHRIFT FUR DIE GESAMTE INNERE MEDIZIN UND IHRE GRENZGEBIETE, (1974 May 15) 29 (10) 410-3.
Journal code: 21730470R. ISSN: 0044-2542.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197411
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19741111
TI [Studies on the mechanism of **nicotine**-induced **lipolysis** in man]. Untersuchungen zum Mechanismus der durch Nikotin induziertem Lipolyse beim Menschen.
CT . . .
Blood Glucose: AN, analysis
Catecholamines: ME, metabolism
English Abstract
Fatty Acids, Nonesterified: BL, blood
Glycerol: BL, blood
*Lipids: ME, metabolism
***Nicotine: PD, pharmacology**
Plants, Toxic
Propranolol: PD, pharmacology
Smoking
Stimulation, Chemical
Time Factors
Tobacco
RN 525-66-6 (Propranolol); **54-11-5 (Nicotine)**; 56-81-5 (Glycerol)

L7 ANSWER 18 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
DUPLICATE 13
ACCESSION NUMBER: 1974:177149 BIOSIS
DOCUMENT NUMBER: PREV197458006843; BA58:6843
TITLE: RESISTANCE OF RAT FETUSES TO **NICOTINE** INDUCED **LIPOLYSIS**.
AUTHOR(S): MOSIER H D JR; CAPODANNO C C; LI I O W; MAGRUDER C S; JANSONS R A
SOURCE: Teratology, (1974) Vol. 9, No. 2, pp. 239-245.
CODEN: TJADAB. ISSN: 0040-3709.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
TI RESISTANCE OF RAT FETUSES TO **NICOTINE** INDUCED **LIPOLYSIS**.
RN 54-11-5 (**NICOTINE**)
57-88-5 (CHOLESTEROL)
51-43-4 (EPINEPHRINE)
51-41-2 (NOREPINEPHRINE)

L7 ANSWER 19 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1974:488968 CAPLUS
DOCUMENT NUMBER: 81:88968
TITLE: Interaction of cholinoreactive systems in the regulation of the metabolism of free fatty acids
AUTHOR(S): Gurin, V. N.
CORPORATE SOURCE: Minsk. Med. Inst., Minsk, USSR
SOURCE: Povysh. Rezistentnosti Organizma Ekstremal'nym Vozdeistv. (1973), 272-9. Editor(s): Mukhin, E. A. Izd. "Shtiintsa": Kishinev, USSR.

CODEN: 28KCA8
DOCUMENT TYPE: Conference
LANGUAGE: Russian
AB Amisyl, which blocks the central M-cholinoreactive system, prevented the increase in the level of free fatty acids in the blood plasma after an i.p.injection of **nicotine**. Atropine or metacin, which block the peripheral M-cholinoreactive system, did not prevent the lipid mobilizing action of **nicotine**, but weakened its hypoketonemic effects. Benzohexonium prevented the increase in blood fatty acid levels induced by arecoline, whereas the central N-cholinoreceptor blockers, pediphen, IEM 506, or prepn. 42, did not. Thus, M-cholinoreactive neurons in the central nervous system play a role in the final link in the transmission of signals to increase **lipolysis**.

L7 ANSWER 20 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1972:414181 CAPLUS
DOCUMENT NUMBER: 77:14181
TITLE: Lowering of serum lipid levels by "masked" nicotinic acid derivatives
AUTHOR(S): Bailey, Denis M.; Wood, David; Johnson, Robert E.; McAuliff, John P.; Bradford, James C.; Arnold, Aaron
CORPORATE SOURCE: Dep. Chem., Sterling-Winthrop Res. Inst., Rensselaer, NY, USA
SOURCE: Journal of Medicinal Chemistry (1972), 15(4), 344-8
CODEN: JMCMAR; ISSN: 0022-2623
DOCUMENT TYPE: Journal
LANGUAGE: English
ST nicotinate deriv serum lipid; cholesterol serum nicotinate; triglyceride serum nicotinate; fatty acid serum nicotinate; vasodilation nicotinate deriv; gastric secretion nicotinate deriv; acyl pyridylmethylamine **lipolysis**; nicotinic hydrazide **lipolysis**; **nicotine** hydroxamate **lipolysis**

L7 ANSWER 21 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1972:509398 CAPLUS
DOCUMENT NUMBER: 77:109398
TITLE: Some aspects of the effect of **nicotine** on plasma FFA [free fatty acids] and tissue triglycerides
AUTHOR(S): Bizzi, A.; Tacconi, M. T.; Medea, A.; Garattini, S.
CORPORATE SOURCE: Mario Negri Inst. Pharmacol. Res., Milan, Italy
SOURCE: Pharmacology (1972), 7(4), 216-24
CODEN: PHMGBN; ISSN: 0031-7012
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Some aspects of the effect of **nicotine** on plasma FFA [free fatty acids] and tissue triglycerides
AB **Nicotine** tartrate (I tartrate) [65-31-6] (3-36 mg/kg, s.c.) increased plasma free fatty acids (FFA), blood sugar, and **lipolysis** in adipose tissue of rats. The lipolytic effects of norepinephrine [51-41-2] or theophylline [58-55-9] were unchanged or slightly diminished by I. The I-induced rise in plasma FFA was inhibited by 5-carboxy-3-methylpyrazole [402-61-9] and by propranolol [525-66-6]. After adrenalectomy I failed to raise plasma FFA. Corticosterone [50-22-6] (15-20 mg/kg, s.c.) restored the effect of I. No elevation of lipids in liver or heart was obsd. after single or repeated treatment with I.
ST lipid metab **nicotine**; fatty acid blood **nicotine**; adipose tissue lipid **nicotine**
IT Blood plasma
(fatty acids of, **nicotine** effect on)
IT Adrenalectomy
(lipid metabolism response to **nicotine** in)

IT Lipids
RL: BIOL (Biological study)
(lysis of, in adipose tissue, **nicotine** effect on)
IT Blood sugar
(**nicotine** effect on)
IT Fatty acids, biological studies
RL: BIOL (Biological study)
(of blood plasma, **nicotine** effect on)
IT Adipose tissue, metabolism
(of lipids, **nicotine** effect on)
IT 50-22-6
RL: BIOL (Biological study)
(lipid metabolism response to **nicotine** and, after adrenalectomy)
IT 402-61-9 525-66-6
RL: BIOL (Biological study)
(**lipolysis** response to **nicotine** antagonism by)
IT 51-41-2 58-55-9
RL: BIOL (Biological study)
(**lipolysis** response to, in adipose tissue, **nicotine** in relation to)

L7 ANSWER 22 OF 24 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
Full Text
ACCESSION NUMBER: 1972:60359 BIOSIS
DOCUMENT NUMBER: PREV197208060359; BR08:60359
TITLE: THERAPEUTIC RISKS.
AUTHOR(S): AMMON
SOURCE: Medizin und Ernaehrung, (1969) Vol. 10, No. 4, pp. 87.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: Unavailable
IT Miscellaneous Descriptors
ABSTRACT BETA RECEPTOR BLOCKERS FATTY-ACIDS **NICOTINE** ALCOHOL LIVER FUNCTION **LIPOLYSIS** BLOCKERS
RN 54-11-5 (**NICOTINE**)
64-17-5 (ALCOHOL)

L7 ANSWER 23 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
Full Text
ACCESSION NUMBER: 1968:493312 CAPLUS
DOCUMENT NUMBER: 69:93312
TITLE: Prostaglandin release from the adrenal gland
AUTHOR(S): Shaw, Jane E.; Ramwell, P. W.
CORPORATE SOURCE: Worcester Found. Exp. Biol., Shrewsbury, MA, USA
SOURCE: Prostaglandins, Proc. Nobel Symp., 2nd, Stockholm (1967), Meeting Date 1966, 293-9. Editor(s): Bergstrom, Sune. Intersci.: Stockholm, Swed.
CODEN: 20AKAU
DOCUMENT TYPE: Conference
LANGUAGE: English
AB In perfused cat adrenal glands, addn. of the secretagog, **nicotine**, to the perfusing fluid did not release materials resembling prostaglandin (I) into the venous effluent. However, the amt. of catechol amines (II) was greater than that released by acetylcholine (III). Hence, the I, PGF1α, is unlikely to be assocd. with the release of II from the chromaffin granules. When Ca++ was introduced into the perfusing soln. to induce release of II, no I-like material appeared in the venous effluent. III was ineffective in releasing either I or II in the complete absence of Ca++. It appeared that I efflux may not be a corollary of release of II, but that Ca++ may be required for III-evoked release of I. Relations between I efflux and **lipolysis** are considered. A I-like material was

extd. from homogenates of rat adrenal glands. ACTH mimicked the effects of III in promoting I formation in homogenates of rat adrenal gland. It appeared that if chromaffin granules contain I, it must be present in bound form. Rat adrenal homogenates had an active lipase which hydrolyzed both tributyrin and Ediol. Release of PGF1α into venous effluents of stimulated adrenal glands was assocd. with efflux of free fatty acids but not of II.

L7 ANSWER 24 OF 24 MEDLINE on STN
Full Text
ACCESSION NUMBER: 68091739 MEDLINE
DOCUMENT NUMBER: 68091739 PubMed ID: 6073319
TITLE: Effect of caffeine, **nicotine** and ethanol on **lipolysis** in human adipose tissue.
AUTHOR: Verdy M
SOURCE: REVUE CANADIENNE DE BIOLOGIE, (1967 Sep) 26 (3) 179-84. Journal code: 8214595. ISSN: 0035-0915.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 196802
ENTRY DATE: Entered STN: 19900101
Last Updated on STN: 19980206
Entered Medline: 19680215
TI Effect of caffeine, **nicotine** and ethanol on **lipolysis** in human adipose tissue.
CT . . .
Adolescent
Adult
Aged
*Caffeine: PD, pharmacology
Epinephrine: PD, pharmacology
*Ethanol: PD, pharmacology
Glycerol: ME, metabolism
*Lipids: ME, metabolism
Middle Age
***Nicotine: PD, pharmacology**
Omentum: DE, drug effects
Stimulation, Chemical
RN 51-43-4 (Epinephrine); **54-11-5 (Nicotine)**; 56-81-5 (Glycerol); 58-08-2 (Caffeine); 64-17-5 (Ethanol)

=> log h

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	154.55	154.76

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-19.40	-19.40

SESSION WILL BE HELD FOR 60 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 12:23:55 ON 27 FEB 2004

10/647,368

WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Friday, February 27, 2004

Hide?	Set Name	Query	Hit Count
		DB=PGPB; PLUR=YES; OP=ADJ	
☐	L17	nicotine and (lipolytic or slenderiz$3)	13
		DB=EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ	
☐	L16	nicotine and (lipolytic or slenderiz$3)	0
		DB=USOC; PLUR=YES; OP=ADJ	
☐	L15	nicotine and (lipolytic or slenderiz$3)	4
		DB=USPT; PLUR=YES; OP=ADJ	
☐	L14	nicotine and (lipolytic or slenderiz$3)	12
☐	L13	nicotine and (lipolytic or slenderiz$3)	12
☐	L12	l11 and lipolysis	12
☐	L11	caffeine same (slimming or cellulite or anticellulite)	77
		DB=USOC; PLUR=YES; OP=ADJ	
☐	L10	caffeine same (slimming or cellulite or anticellulite)	0
☐	L9	nicotine same (topical or cosmetic or dermatological)	34
		DB=USPT; PLUR=YES; OP=ADJ	
☐	L8	US-5158771-A.did.	1
		DB=EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ	
☐	L7	nicotine same (topical or cosmetic or dermatological)	32
		DB=USPT; PLUR=YES; OP=ADJ	
☐	L6	L5 not l3	101
☐	L5	nicotine same (topical or cosmetic or dermatological)	113
☐	L4	l3 and (nicotine near5 (concentration or percent or amount))	30
☐	L3	(nicotine and (gel or cream or ointment or lotion or emulsion or balm or spray or patch or stick)).ti,ab.	36
☐	L2	L1 and (gel or cream or ointment or lotion or emulsion or balm or spray or patch or stick)	918
☐	L1	nicotine and (topical or cosmetic or dermatological)	1000

END OF SEARCH HISTORY